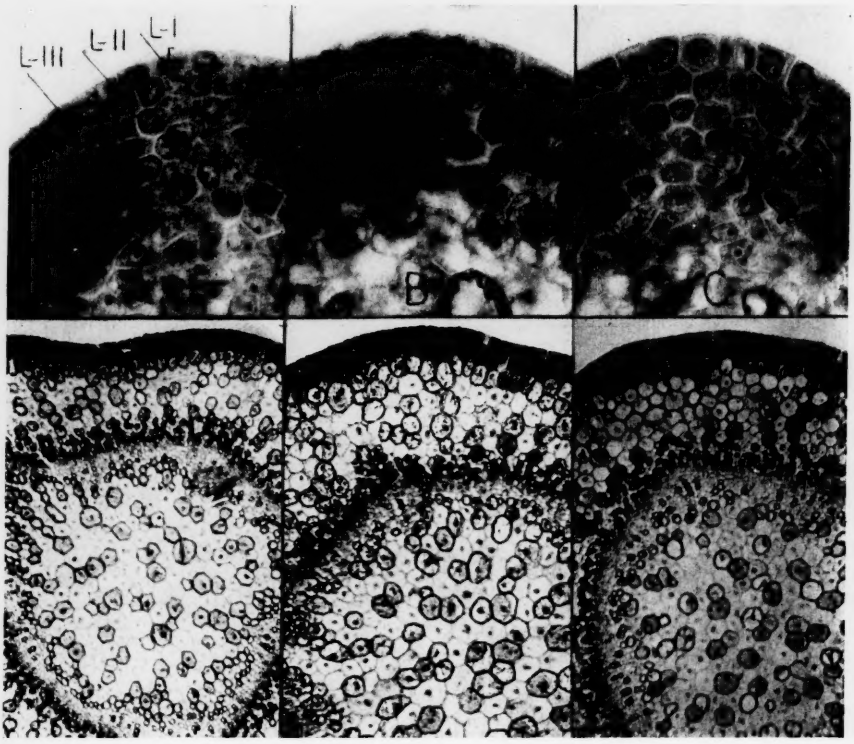
The Journal of HEREDITY



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The Stockholm Congress

Lysenkoism Now Official in the Soviet Union

A New Theory of Otosclerosis

The Black Baby in Folklore and Science

Tasteblindness and Diabetes

VOLUME 39 AUGUST, 1948 NUMBER 8

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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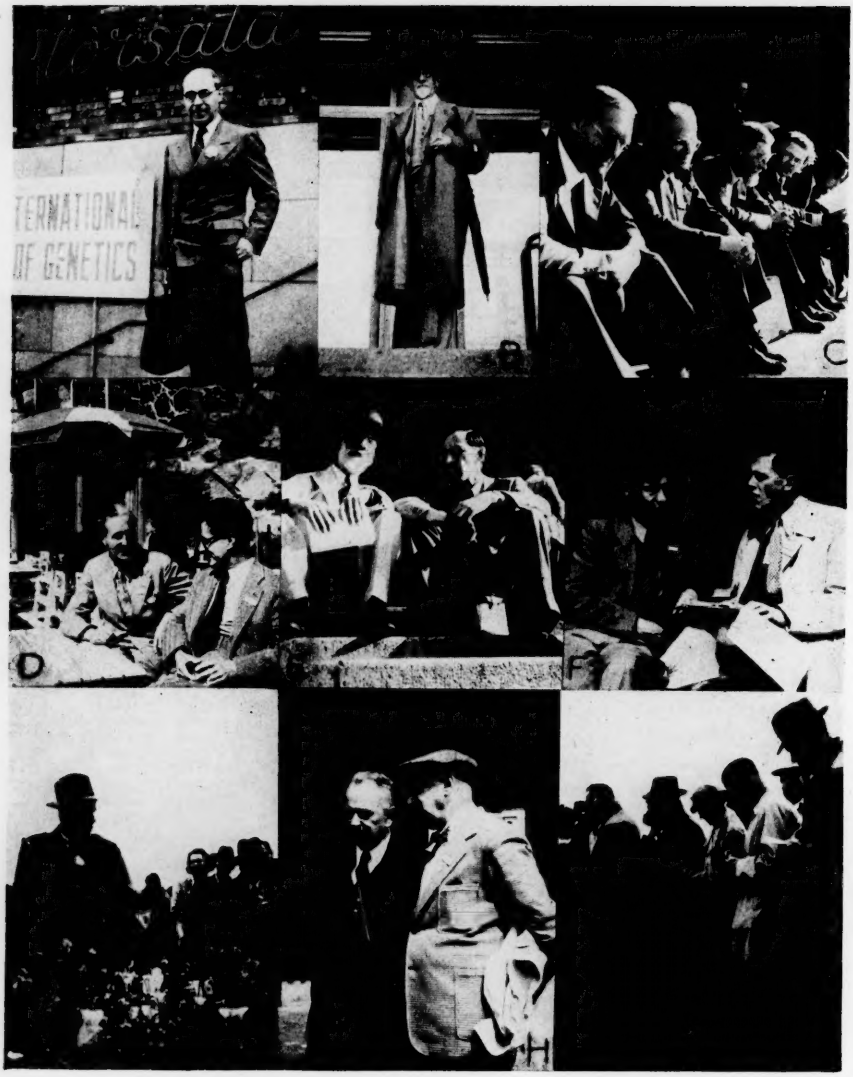


GENETICISTS IN THE SCHEME OF STOCKHOLM

Figure 1

A—Congress President Dr. H. J. Muller in front of the Medborgarhuset, where the Congress met. *B*—Dr. E. Tschermak von Seysenegg, the only surviving member of the trio who in 1900 re-discovered Mendel's paper. *C*—An informal "Genetics Conference" on steps of Medborgarhuset. Present are H. Lamprecht (Sweden), M. Demerec (U. S.), M. J. Sirks (Netherlands), M. J. Heuts (Belgium), and C. C. Tan (China). *D*—C. G. Darlington and J. L. Fyfe (England) relax between meetings. *E*—R. A. Fisher (England) and Boris Ephrussi (France) on the steps of the Medborgarhuset. *F*—German delegates A. Lang and G. Melchers at Lund during the South Sweden pre-congress tour. *G*—Arne Muntzing in the field of Svalof. *H*—Pre-congress demonstration viewed by P. S. Hudson (England), A. Ernst (Switzerland), E. Schiemann (Germany), J. Bustarret (France), R. Goldschmidt (U. S.), W. Black (England), and J. G. Davies (Australia). *I*—Th. Dobzhanski and Richard Goldschmidt snapped during the pre-congress tour in South Sweden.

THE STOCKHOLM CONGRESS

Nearly 600 Geneticists from 37 Countries Attend First Post-War Meeting

AFTER an interval of nine years during which the civilized world came close to accomplishing its self-destruction, a conclave of the geneticists of the world has amply proved that not every international conference need end in discord, rancor, and disunity. The Eighth International Congress, which met at Stockholm from July 7 to 14, set an example of reasonable exchange and discussion of views that were by no means always concordant, which more pretentious bodies whose agenda are concerned with the intercourse of nations in concert might do well to emulate. During those dark hours through which the world has just passed, when it seemed that the only light was shed by the lurid holocaust of battle, we now learn, at Stockholm and elsewhere this summer where scientists of various disciplines are convened, that the lamp of learning, albeit obscured, was never extinguished. It continued to burn in varying degrees of intensity in scientific laboratories everywhere. Well—almost everywhere.

That it was snuffed out in some places is revealed in the names that failed to answer when the roll was called. Among the twenty countries which were represented at Edinburgh in 1939 and which were absent at Stockholm in 1948 we may note as significant Burma, Ceylon, Estonia, Greece, Malaya, Netherlands East Indies, Palestine, Roumania; all these were, or are presently, engulfed by war. That there is some room for optimism is indicated by some of the names that, surprisingly, answered "present." Japan was there, with one delegate. Germany sent eleven representatives, at least two of whom, Hans Stubbe and Paula Hertwig, were from the Russian zone. There were a dozen-odd others from eastern Europe, Hungary, Poland, Czechoslovakia, Bulgaria, Jugoslavia. China, which has known no peace for twenty years, was there. Austria which in 1939 was not represented in its own name sent a delegation of three, headed by a venerable, delicate old man with a pointed beard, gnarled arthritic hands, and the beautiful manners of a vanished tradition, Professor Tschermak von Seysenegg of Vienna, the last of that famous triumvirate who rediscovered Mendel's laws. Present too was Pakistan, one of the new nations on the earth.

Genetics in the 1948 Scheme

The most noteworthy absentee, at this congress as at the last, was Soviet Russia. To the invitation issued by the organizing committee the reply came back through diplomatic channels from the Russian Academy of Science: "The Russian geneticists are too busy to leave their work." The heartbreaking and tragic schism that this answer acknowledges, if at all only by its curtness, was placed squarely before the opening plenary session of the congress by its president, Dr. H. J. Muller. In his scholarly and brilliant presidential address that ranged far and wide on the ramifications of "Genetics in the Scheme of Things," Dr. Muller devoted a lengthy section to a review of some of the events in the genetics battle and to a bitter denunciation of the Soviet trend toward anti-science. When the Seventh Congress was still scheduled to be held at Moscow, he revealed that one of the conditions which the Russians wished to impose was that no papers be read touching on human genetics, a condition which must be unacceptable to any conscientious man of science.

With sorrowful tones appropriate to the pronunciation of the names of comrades who have fallen in the struggle for what they believe right, Muller read the honor roll of those martyred Russian geneticists, headed by the illustrious N. I. Vavilov, who have disappeared without a trace. In the strongest terms, he

decried a "science" which condemns as treasonable any results which deviate from what are ordained from on high as the conclusions to be reached. Reaffirming his adherence to the principles of the *Geneticists' Manifesto* (promulgated in 1939 at the conclusion of the Seventh Congress—and not an official document of the Congress—as a formulation of the highest hopes which the science of genetics could offer a sorely troubled world), President Muller stressed that the betterment of the social and economic lot of mankind remains a *sine qua non*. He stressed emphatically that the subversion of the freedom and dignity of the individual to a totalitarian regime in the name of this program of betterment is too high a price for achieving it.

The assembled geneticists, some 600 strong, representing three dozen countries, listened intently to these strong words. For the majority who had watched in silence for fifteen years while their science was perverted by polemics and rent by schism it was heartening to hear at last a full-dress, official rejoinder on behalf of classical mendelism. The pleasure which the majority took in these sentiments, expressed in hearty applause in the auditorium and in more articulate approval in the corridors afterward, was not shared unanimously. Following his introductory lecture on human mutation rates, J. B. S. Haldane put his manuscript aside and solemnly "apologized" for having conducted the research which he had just elucidated "under the inspiration of a philosophy which our president has been at such pains to take to task." It was not until a week later that any other divergent view was voiced. At the closing session of the Congress, after Muller had turned over the chair to the Vice-President, Federly of Finland, one of the Polish members rose and in mild and undramatic fashion made a brief statement, the purport of which was that some delegates present wished to go on record as taking exception to that part of the presidential address which referred to Russia. Federly declared that this statement would be incorporated in the official proceedings. There the matter ended. Those who had hoped to see the differences between Mendelian genetics and Lysenko genetics thrashed out in an open forum of experts were sadly disappointed.

Genes, Spores, and Blood (Rh)

If the issue which President Muller had so squarely posed at the opening session still remained unjoined at the Congress' conclusion, the arduous week of working sessions amply demonstrated which school of thought guided these geneticists in their laboratories back home. For six and a half hours a day for seven days, in the two hundred papers read and discussed, the order of business was the present state of the classical genetics of Gregor Mendel. A random geographical sampling of the program reveals both the progressivism and the orthodoxy of modern-day genetics. For example:

Iceland (A. Love)—Mutations at the crater of Hekla in eruption.
Poland (E. Malinowski)—Mutations of Pleiotropic alleles in petunias.
Brazil (C. A. Krug)—Genetics of coffee.
Finland (E. Suomaleinen) — Parthenogenesis and polyploidy in weevils.
Czechoslovakia (F. Hercik) — A radiation method for determining the number of genes in spores of Rhizopus Nigricus.
China (C. C. Tan)—Seasonal variations of color pattern in lady-bird beetles.
England (Charlotte Auerbach) — Chemical mutagenesis.

Mendel, Bateson, Morgan, et al might be surprised, and would surely be pleased, at the strides made by genetics, but they would have no trouble recognizing the infant science they once knew so well.

The calm and measured pace of the working sessions suddenly quickened at one point, and for the space of a morning words were bandied with heated eloquence, arguments were pressed with the ardor of partisanship, and a little blood was spilled. The protagonists were, on the one side, Race and Fisher, with a brilliantly-timed assist from Bentley Glass; on the other side, aggressive clever, competent, and not at all phased by the role of *uno contra mundam*,

stood Alexander Wiener. The blood that was spilled was Rh blood, and the squabble, of course, was how to classify it. Glass presented a case (in a negro family) that seemed in his view to be indisputably a crossover. Of the four possible explanations of the child's phenotype, i.e. mis-typing, mutation, illegitimacy, or crossingover, he adduced evidence for the elimination of all but the last. Which being the case, there was no other conclusion possible than to accept the three-locus theory of Fisher-Race. Weiner promptly rose and questioned the logic which had excluded the possibility of illegitimacy. The configuration of the fingerprints of the child and the putative father, which Glass had assumed to be sufficiently rare to be conconvincing presumptive evidence of paternity, Weiner asserted occurred in negro populations with a high degree of frequency. Then all of the principals in turn took the floor. When the chairman's gavel finally silenced all contending voices and declared the session to be closed, the confusion of a double nomenclature and of two incompatible genetical theories still remained in the domain of blood group genetics.

Space does not permit a detailed account of the enormous amount of genetics which was brought to this congress from all over the world. Nor does fairness allow this brief report to single out for special mention any individual papers. These will all be carried in full in the *Proceedings,* which will be published at some unspecified future date. However, certain of the official acts of the congress in plenary session are of interest to all geneticists. It was decided to affiliate with the FAO (for one year, provisionally) on a project for the cataloguing of genetical stocks throughout the world. It was decided to hold the next congress in 1952, and to hold future congresses at four-year instead of five-year intervals. Several invitations for the next congress were received, notably from the United States and from Italy. Indications seemed to be that the 1952 congress will be held at Rome, although the final decision has not been taken.

Swedish Hospitality

The smooth functioning of this congress is a tribute to the painstaking care of the organizing committee and to the able administration of the Secretary General, Professor Gert Bonnier. The Swedish hosts spared neither hard work nor money to ensure that the physical surroundings as well as the social concommittants of the working congress should be of the highest order of convenience, amiability and hospitality. Aside from the innumerable courtesies of the individual Swedish geneticists to the foreign visitors, on an official level the hospitality was munificent. Sweden put up 40,000 kronor (about $11,500). The city of Stockholm donated Medborgahuset, the beautiful municipal structure which housed the congress, and gave in addition an enormous official reception to the entire congress. Other receptions where libations of hospitality were offered with equal generosity were held for the congressists by the University of Lund and the University of Uppsala. The voluminous printing in connection with the congress was undertaken without pay by the printing firm of Albert Bonnier. The Eighth International Congress of Genetics, a happy reunion after a long and painful separation, would have been a scientific success under any circumstances. It has become, thanks to the fortunate blending in its Swedish hosts of the twin virtues of organizing ability and hospitality, a model for all future congresses to emulate.

MORTON STARK

". . . The Russian Geneticists Are Too Busy to Leave Their Work . . ."

The New York Times *for August 19 carried the following news story of recent developments in the genetics controversy in the U.S.S.R.:*

After more than a decade of struggle, T. D. Lysenko, noted Russian plant breeder, has emerged victorious over Soviet geneticists who have long opposed his theories, articles in Pravda from Aug. 3 to 11 report.

A decision of the Central Committee of the Communist party of the Soviet Union now apparently assures that the "Western and bourgeois" genetic theories of Gregor Mendel and Prof. Thomas Hunt Morgan are to be banished from Russian laboratories and schools, to be replaced by work based on the theories of Professor Lysenko and his predecessor plant breeders, particularly Ivan V. Michurin.

Professor Lysenko's triumph came after an eight-day conference, July 31-Aug. 7, of the All-Union Academy of Agricultural Science held in Moscow. Professor Lysenko opened the meeting with a major address reiterating his view that plants and animals can transmit acquired characteristics and scoffing at geneticists who claim to have disproved this.

He denounced academic Soviet geneticists, including I. I. Shmalhausen, B. M. Zavadovsky, A. R. Zhebrak, N. P. Dubinin and others for accepting and teaching these Western theories and rejecting Lysenko's views.

Several days of debate followed during which the overwhelming majority of participants backed Professor Lysenko.

Several of Professor Lysenko's opponents spoke up vigorously against him. Dr. Zavadovsky accused Dr. Lysenko of misrepresenting the position of those he attacked and of demanding uncritical acceptance of his views even when they were not supported by experimental evidence. Professor Zhebrak cited a large number of experimental results supporting the chromosome theory of inheritance. Docent S. I. Alikhanyan of Moscow State University pleaded that Soviet science be allowed to proceed on all possible fronts and not be confined to the theories of one school only. Academician Shmalhausen denied Lysenko's accusations of accepting idealistic philosophic concepts and declared that his theories were in full consonance with Marxian materialism.

At the end of the discussion, Professor Lysenko revealed that his introductory address had been read and approved by the Central Committee of the Communist party of the Soviet Union. With this announcement opposition faded out. Academician P. M. Zhukovski, Docent S. I. Alikhanyan and I. M. Polyakov announced their complete submission to Lysenko's views.

The Academy of Agricultural Science then unanimously adopted a letter addressed to Premier Stalin in which it declared that Soviet agricultural science was the most advanced in the world. It also adopted a resolution that called for the rewriting of university textbooks and the revision of courses in biology and related sciences so as to remove all traces of foreign geneticists' views and to bring all teaching in conformance with the Lysenko-Michurin position.

✦

From the *New York Times* for August 25:

Prof. Anton R. Zhebrak, internationally known Soviet geneticist, has written a letter to Pravda, official Communist party organ, in which he announces his submission to the officially endorsed "party line" views on the genetics of T. D. Lysenko, his opponent in a fifteen-year struggle of "Marxist" versus "bourgeois" concepts of heredity.

This battle, analogous to the Nazi differentiation between "pure Aryan" and "non-Aryan" science, which led to the exile of scores of Germany's top scientists and contributed greatly to Germany's defeat, has already led to the purging of scores of leading Soviet geneticists and biologists, including the liquidation of Prof. Nikolai Vavilov, universally recognized as one of the world's top scientists, and many others. * * *

Professor Zhebrak's letter of recantation, addressed to the editors of Pravda, states that formerly the Communist party had permitted both pro-Lysenko and anti-Lysenko views to be voiced. However, he adds, since the Central Committee of the Communist party had approved Lysenko's views, "I, as a party member, do not consider it possible for me to retain the views which have been recognized as erroneous by the Central Committee of our party."

Professor Zhebrak went on to assert that his views had never been identical with those of Mendel, Morgan and other Western geneticists, and that he had fought those ideas in classical genetics that were opposed to dialectical materialism. He also asserted that he had long been a follower of Ivan Michurin and other Soviet plant breeders, and that as a result of following their techniques had made significant scientific contributions. * * *

Pravda's editors also assail Zhebrak's claim of having made valuable experimental contributions, asserting that he and his laboratory had contributed "nothing of value to Soviet agriculture."

The editors conclude by accepting Zhebrak's recantation, but warn him that his sincerity in accepting the views he had fought so long would be judged on the basis of his future work. * * *

HEREDITY AND ENVIRONMENT IN OTOSCLEROSIS

ELAINE W. AMIDON*

OTOSCLEROSIS is a type of deafness that affects about 1,500,000 persons in the United States. The large number of persons affected makes its study important. In addition, it seems to belong to that interesting class of diseases and defects in which a knowledge of hereditary pattern is essential to proper medical diagnosis, prognosis and treatment. In turn, medical research fills in the gaps in our knowledge to build a more complete picture of the hereditary component of otosclerosis.

Actual loss of hearing from otosclerosis begins when the stirrup, one of the tiny connecting bones in the middle ear, becomes fixed to the oval window of the inner ear. Degree of hearing loss depends on degree of fixation of the stapes (stirrup) and differs greatly from family to family; and, to a lesser degree, differs within the same pedigree. However, this fixation of the stapes is the result of a process initiated earlier.[9] The capsule enclosing the inner ear, about as big as a cherry, is composed of exceptionally hard bone, and is sometimes referred to as the petrous capsule. It is here that the otosclerotic process usually begins. Nager[11] says that it starts by localized resorption of old bone by lacunar erosion, and replacement of this by immature, web-like bone tissue containing much cementum. Guggenheim[7] says that surrounding the focus of otosclerotic bone is usually an area of decalcification or halisteresis. This area of "preparedness" finally becomes a cavity filled with osteogenic marrow where new abnormal bone forms, thus increasing the size of the original focus. It is obvious that the abnormal growth does not have to be great to fix the stapes to the oval window, since the window itself is about the size of a small pearl.

Distribution of Otosclerosis

Otosclerosis has been observed in all civilized countries; Nager[11] reports that in some regions it seems to have a higher frequency than in others. Frequencies are not known for other than the white and negro races. Bunch[2] says it is rare in negroes, and his finding is confirmed by Guild.[9] Authorities agree that the most common age of onset is between sixteen and thirty. Actual deafness from this cause rarely appears under fifteen. Davenport[3] analyzed a large number of pedigrees. He found the frequency of otosclerosis to be about twice as high in females as in males. In another study, Davenport[3] found that in a sample limited to patients under thirty, the proportion changed from 66 ♀ : 34 ♂ to 54 ♀ : 44 ♂. Nager[11] in a series of private patients found the proportion eight to one.

Histologic Otosclerosis

Histologic otosclerosis is the existence of otosclerotic processes in the petrous capsule even when the changes are not extensive or not in the right area to cause clinical, measurable, deafness. This concept of otosclerosis is comparatively recent. Guild[9] reports a series of 1161 autopsies in which human temporal bones were studied. This constituted a random series as far as deafness is concerned. His results are summarized in Table I:

TABLE I. Guild's autopsy series.

Sex	Normal	Histological Otosclerosis	Percent Otosclerosis
		WHITE	
Male	368	24	6.1
Female	173	20	10.3
		NEGRO	
Male	391	4	1
Female	180	1	.52
TOTAL	1112	49	

*Affiliated with the Genetics Division, Dept. of Plant Sciences, Syracuse University, Syracuse, N. Y.

Guild[9] also points out that in this series more active lesions were found in the younger age group, especially under twenty. While it has been considered by some authorities that atrophy of cochlear nerve fibers within the inner ear also was involved in otosclerosis, Guild[9] found in this study no correlation between the two kinds of deafness. On the basis of this study Fowler[4] estimates that to the 1½ million persons in the United States who are actually deaf from clinical otosclerosis must be added about ten million who possess otosclerotic foci.

Hypotheses Concerning the Heredity of Otosclerosis

Davenport and his co-workers, analyzing a series of pedigrees, taken in part from the literature on otosclerosis and in part collected by them, reached some negative conclusions and advanced an hypothesis. The theory of a single dominant was discarded because a number of cases are known where two normal people have had otosclerotic children (Nager[11] found, more recently, a family history in only 52 per cent of his patients). A single recessive inheritance was discarded because the average number of otosclerotic children in families where both parents have otosclerosis was 70 per cent, instead of the expected 100 per cent. Sex-linked dominance was discarded for reason cited above, and a sex-linked recessive inheritance was discarded because there are more affected females than males. The actions of two recessive and of two dominant genes were discarded because of inconformities between expectation and data. They finally adopted the hypothesis that the conditioning of otosclerosis by one sex-linked dominant gene and one autosomal dominant gene would account for the observed data. Dr. Davenport says otosclerosis is "determined by two dominants, a factor *X*, which lies in the sex chromosomes, and a factor *A*, which lies in one of the autosomes." Although he states that otosclerosis develops under conditions that favor it, his theory is actually based on pedigree analysis of demonstrated, not inherent, otosclerosis.

The statistical basis of this conclusion is worth examining, since it is based on the largest series in the literature of pedigrees studied, including pedigrees in which only the offspring were affected; pedigrees in which only parents are affected; those in which only the mother is affected; and pedigrees in which only the male parent is affected. One of Davenport's most important conclusions, on which he based his theory was this: "When the mother only is affected, affected sons will be common, but when the father only is affected, affected sons will be less common. According to table 6, when the mother only is affected, 45% of the otosclerotic offspring of known sex are sons. When the father only is affected, 31% of the otosclerotic offspring of known sex are sons."

Table II (from the article by Davenport and his co-workers) is shown, to demonstrate what Dr. Davenport meant, and what other meaning may more easily be drawn from the same figures.

Considering only the figures collected by Dr. Davenport, it can be observed that there is no significant difference in the *proportion* of the otosclerotic males in these two tables. There is a real difference in the proportion of the total otosclerotic males because in the sample wherein the father only is affected there is a greater proportion of females. But

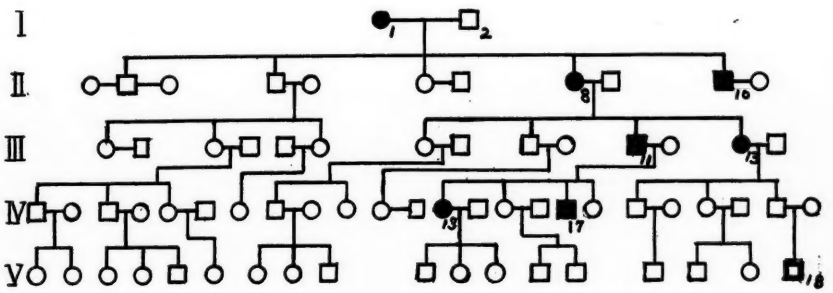
TABLE II. Davenport's Table VI, a summary of actual findings in fraternities containing affected children, pp. 163-164.

SUMMARY OF FRATERNITIES IN WHICH THE FATHER ONLY IS AFFECTED—Children over 30.

	Sex	Number	Otosclerotics Number	Otosclerotics Per cent
11 original fraternities collected by Dr. Davenport & colleagues	male	21	4	19
	female	39	15	38
12 fraternities from literature	male	22	10	45
	female	24	17	71

SUMMARY OF FRATERNITIES IN WHICH THE MOTHER ONLY IS AFFECTED—Children over 30.

	Sex	Number	Otosclerotics Number	Otosclerotics Per cent
11 original fraternities collected by Dr. Davenport & colleagues	male	15	3	20
	female	24	10	42
14 fraternities from literature	male	37	22	59
	female	33	17	52



FOUR GENERATIONS OF OTOSCLEROSIS

Figure 2

In this family otosclerosis appears to be inherited as a simple dominant character. Over a period of a good many years, several hypotheses have been suggested as to the mode of inheritance of this form of deafness. Different genes for otosclerosis may exist in different families, but it is suggested that these disparities are due to th action of a dominant gene with rather low penetrance. Hence many potentially otosclerotic individuals are not deaf. The preponderance of otosclerotic females would be accounted for on the assumption that the penetrance is greater in the female sex because crises in calcium metabolism, such as pregnancy, stimulate otosclerotic changes in the ear.

this is not a large enough difference to be significant, even without the greater proportion of females. To arrive at the figure that 45 per cent of the otosclerotic offspring of affected mothers are sons, while only 31 per cent of the otosclerotic offspring of affected fathers are sons, the totals from the literature must be added in, altering the proportions entirely. This, in effect, determines the entire conclusion of the article. Is it valid to determine the conclusion of an investigation almost entirely on figures collected by a number of different people at widely separated dates, and no doubt with different standards; and to ignore the different implications of the figures from the original work done?

This part of the discussion could almost be omitted on the basis of the conclusions drawn by Guild regarding the heredity of otosclerosis. He states: "The facts observed in the present material render invalid all conclusions with respect to dominant and recessive genes that have been drawn from clinical studies of the inheritance of otosclerosis. To obtain a sufficient basis for a reliable determination of the hereditary factors in otosclerosis it would be necessary to examine histologically the temporal bones of all members of many family groups; independent of their acuity of hearing. Only thus can the presence or absence of otosclerosis be determined."

However, such an enterprise is far in the future. The above pedigree, although only of clinical otosclerosis, is of some interest. In this particular pedigree its inheritance appears to be simply dominant. The pedigree includes a fifth generation, but all the members are too young to demonstrate the gene. I-1 was extremely deaf by middle age. Several children born to her died in infancy, and are not indicated on the pedigree. II-8 was also severely deaf by middle age. II-10 was only moderately deaf at his death, around thirty. III-11 is moderately deaf, but the female, III-13, is severely deaf, yet they are only a year apart in age. In the fourth generation the trend for the females to be more severely deaf is reversed, IV-13 being moderately deaf, while IV-17 is severely deaf. It is an interesting point that the female in this generation suffered little or no hearing loss following preg-

nancy — and made a distinct effort to take extra calcium and vitamin D at this time. The male, on the other hand, was at one time during adolescence on a low calcium diet.

Otosclerosis Considered As a Possible Irregular Dominant

It seems possible that otosclerosis is an irregular dominant, with strong penetrance in some pedigrees, weak in others. H. B. Glass[5] mentions hemolytic jaundice and a non-sex-linked type of hemophilia as being human defects of this nature. These types of inheritance, he points out are with difficulty differentiated from those due to the interaction of two or more genes.

Consideration of otosclerosis as an irregular dominant, whose penetrance is affected by environment to some extent might explain why women display the defect more frequently than men. Men and women having the same genotype may show a different phenotype, as is quite consistently the case with sex-influenced characteristics like baldness. In this situation the gene would tend to be dominant in men and recessive in women. The internal environment of men and women does vary in important respects, and with respect to that form of internal balance affecting the bony structure, it varies considerably.

Otosclerosis shows a marked tendency to become worse following pregnancy and lactation. While Nager[11] minimizes this effect he does say that 79 out of a series of 164 otosclerotic women who had borne children said they had noted an increase in deafness at that time. In Dr. Davenport's series of pedigrees the age group under thirty showed a sex-distribution of otosclerosis approximately 1:1, while the distribution for all ages was about 2 ♀ ♀ : 1 ♂ ♂. This is interpreted by Davenport as indicating that the full potentialities of the daughters have not developed under thirty. Is it not equally possible that the potentialities for damage due to this gene are not developed in the sons at any age as fully as in the child-bearing women?

Theories on the Pathology of Otosclerosis

That there is any definite abnormality of any gland in otosclerotics, has never been proven. Attempts have been made to incriminate the thyroid, the parathyroid and the pituitary glands. Robb[12] gave pituitary and parathyroid extract to dogs and found histologic changes in the ear similar to those in human otosclerosis. However, animals in the experiments showed widespread abnormal changes in bone structure, while otosclerosis is a localized affair.

Benesi[1] believes otosclerosis may be regarded as a continuation of growth beyond normal limits, slow at first, accelerated by secondary factors such as puberty, pregnancy, violent exercise, disease, or by any conditions which are accompanied by high temperatures and disturbed general metabolism. Hagen[10] concurs in this view, adding faulty diet as a factor. Gottlieb[6] believed the hereditary factor could be ignored entirely and says otosclerosis is due to persisting environmental factors, such as nutrient and toxic disorders.

As was demonstrated by Guild's work on histological otosclerosis, alterations in the bony structure of the ear start long before deafness appears. A certain threshold is reached—and slowly the individual begins to lose hearing. Is it not possible that a threshold must be reached in the internal chemistry of the body before these alterations start? Nager believes that otosclerotic foci start by means of the lack or presence of some substance in the circulation. This would agree with a theory of irregular dominance in the pattern of inheritance.

Glass[5] says of this type of inheritance: "There is quite generally a minimum concentration which a substance determining a reaction must reach before it can act—this is the threshold. Together with the fact that, from individual to individual within the same genotypic class, there is normal variation due to "chance," the existence of thresholds is responsible for a considerable part of the confusion and difficulty in our think-

ing past and present about heredity.... Individuals of the same genotype may and often do differ in phenotype."

Fowler, considering that one in twelve members of the white race have lesions in the petrous bone, suggests that otosclerosis be considered more as an unexplained morphologic variation, due to some influence outside the petrous bone rather than to think of it wholly as a disease. In any other location similar small lesions would rarely cause disease.

Possible Activators of the Gene

The question remains as to what internal mechanism initiates the otosclerotic process in the ear. A very important point here is that the actual onset of otosclerosis is not by over-growth of bone, but by a dissolving out of calcium previously contained in the bony cells. Later comes the replacement and overgrowth, according to Nager.[11]

The parathyroid gland secretions cause an increase in the blood calcium by resorbing bone. Williams[13] says that "While the usual effect of the hormone is to increase osteoclastic activity and the removal of calcium from the bone, it sometimes causes the formation of osteoblasts which reverse this process." This is very suggestive since otosclerosis is a tearing down of bone and a rebuilding.

Under normal conditions, the calcium level of the blood is maintained within a narrow range by the internal mechanisms of the body. The balance is held, notwithstanding normal fluctuations in the supply of calcium and vitamin D.

Otosclerosis frequently has its onset or grows more severe following times when the calcium balance has been under some stress; for instance, following adolescence—when the child grows at a terrific pace for a time. The parathyroid secretions, normally increasing to maintain the calcium balance during a time when the body found calcium in short supply, might be the chemical messengers that initiate the otosclerotic processes. The difference between otosclerotics and normals might be just a difference in the point from which calcium was resorbed from the bones when needed. Two people with the same genotype would then have a different phenotype with regard to otosclerosis if their diet was different.

Preventive calcium therapy has been tried in a few cases by Fowler[4] on the children in otosclerotic families, and so far the children do not display the trait. Whether they would have, without treatment, is not certain. Guggenheim[8] has had some success in arresting otosclerosis by treatment with calcium, phosphorus and vitamins.

Tentative Conclusions

It appears reasonable to consider that otosclerosis may be an irregular dominant with variation in penetrance from two causes, one hereditary; one environmental: sex influenced, but not sex-linked.

1. Actual difference in the hereditary pattern of the otosclerotic focus—as in other genotypes—for instance, pattern baldness.
2. Environmental influences, acting through the endocrine system, to alter the phenotype.

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TASTE-BLIND IDENTICAL TWINS

With Diabetes and Other Striking Pathological Characteristics

GABRIEL SEGALL, M.D.*

THE importance of studies of twins, in evaluating the role played by heredity in human variation, has long been evident to workers in biology and medicine. An extensive literature on the similarities and differences of identical and fraternal twins is already at hand. The pair of twins to be described in the present paper seems worth adding to this literature for several reasons. The extensive clinical and hospital records covering virtually their entire lives to date provides descriptive material not often available on so broad a basis in twin studies. Both members of this pair experienced a number of their symptoms nearly simultaneously in occurrence, suggesting specific hereditary determination of these abnormalities. Furthermore the twins are the only non-tasters for phenylthiocarbamide ("PTC"), and the only diabetics, in their family. This is of primary interest to the author, who, with Dr. M. C. Terry, previously noted a statistically significant association between taste-blindness and diabetes in human populations.[1]

General Description of the Twins and Their Family

The twins were born on November 21, 1927. Their photographs (Figure 3), taken at the time of preparation of this paper, show them at the age of 20 years, 5 months. Table I summarizes qualitative observations of genetic interest on their father, mother, two brothers, and the twins themselves.

The twins are identical with respect to the erythrocyte characteristics *O, MN,* and Rh_o, and with regard to hair color, eye color, and taste-reactions. In four of these characteristics, blood group, hair color, eye color, and taste-reaction to PTC, the twins share qualitative differences from one or both of their brothers, and from one or both of their parents. Only a single placenta is recorded as having been delivered at the birth of the twins. These observations, together with the other similarities to be detailed below, leave small basis for doubt that the twins developed from a single fertilized egg.

The inclusion of the compound *brucine* in the "taste" column of Table I requires a word of explanation. Brucine has been the subject of unpublished studies by Beadle and Mitchell, and of a Master's thesis at Stanford University by Barrows.[2] These workers have observed sharp differences in humans, in the ability to taste this compound, comparable to but independent of the PTC reaction, and have marshalled some evidence on the heritability of brucine taste-blindness. It is sufficient here to note that the twins and all their family are "tasters" for brucine. The twins seemed to react somewhat more slowly than do

TABLE I. Descriptive data.

Individual	Blood Groups			Hair and Eye Color		Taste	
	A-B-O	MN	Rh_o	Hair	Eyes	PTC	Brucine
Father	A	MN	+	Dark Brown	Hazel	Taster	Taster
Mother	O	MN	+	Dark Brown	Blue	Taster	Taster
Son, 26 yrs.	A	MN	+	Blonde	Blue	Taster	Taster
Son, 32 yrs.	A	MN	+	Dark Brown	Brown	Taster	Taster
Twin *X*	O	MN	+	Med Brown	Blue	Non-taster	Taster
Twin *Y*	O	MN	+	Med Brown	Blue	Non-taster	Taster

*Associate Attending Physician, Cedars of Lebanon Hospital, Los Angeles, California. The author is indebted to Doctor Morris H. Nathanson, Attending Physican, and to Doctor S. Z. Goodman, Attending Physician (Diabetic Service), of the Cedars of Lebanon Hospital, for the use of their records. Blood tests were performed by the Clinical Laboratories of Doctors Zeiler, Hammack and Maner. The electro-encephalographic studies, and their interpretation, by Dr. N. A. Bercel, are gratefully acknowledged. Doctor R. D. Owen, of the California Institute of Technology, contributed suggestions relative to the conduct of the study and its publication.

most brucine tasters.

The parents were both born in Russia, and are Jewish, a fact noted here because of the higher incidence of diabetes in Jewish populations. Neither the father nor the brothers of the twins have any known disability or abnormality relevant to this report. There is no record of diabetes, nor of any other pathology comparable to that encountered in the twins, among other blood relatives of the parents. The mother, however, in a very recent examination occasioned by the preparation of this paper, did display a peculiar neurological anomaly, comparable to one of the abnormalities shared by the twins. This has been verified by Dr. Theodore Rothman, a neuropsychiatrist. The anomaly of the mother is tentatively diagnosed as a diffuse lesion of the brain, mainly involving the cerebellum. The diagnosis is based on the observation of: 1) left adiadochokinesis; 2) impairment of cerebellar posture-holding function on the left side; 3) impairment of gait, with a tendency to walk to the left; 4) dysmetria of the left hand in the finger-to-nose test; and 5) diminution of the biceps reflex on the left side, and absence of the Achilles-jerk reflex of the left side. Her electroencephalogram shows no abnormalities.

The mother was 35 years old when the twins were born. There was some evidence of toxemia in this pregnancy; her ankles were swollen during the last week of the pregnancy. She had suffered from heart disease for several years prior to the pregnancy; this has been diagnosed repeatedly as mitral stenosis with enlargement of the left auricle.

Readings with the Collens oscillometer for the mother and the twins are summarized in Table II. Although not as extreme as the twins, the mother also shows abnormalities indicated by oscillometer readings well below the normal range. This is evidence of a peripheral vascular disturbance in the mother comparable to, but not as severe as, that found in the twins. The twins will be described in this respect later in this paper.

Birth and Infancy of the Twins

The twins were born after a term of pregnancy of 8½ months, and after 1½ hours of labor. Rupture of the amnion occurred with the onset of labor pains. Twin X was delivered first; Y was born five minutes later. Both were born with head presentation. Weights and measurements at birth were:

Twin X:	Weight 5 lbs. 6 oz.	Length 17″
	Head 12″	Chest 10-½″
Twin Y:	Weight 5 lbs.	Length 16-¾″
	Head 12″	Chest 10″

No abnormality was noted in either child after delivery; no jaundice, head injury, fever, convulsions, delerium, or coma. A single placenta was delivered ten minutes after the birth of the second twin. No record was made of the structure of this placenta.

The first tooth of each twin appeared when they were nine months old. They began to talk at about one year. They took their first steps at the relatively late age of 15 months. The following symptoms developed at the age of about five months: Both of the twins became easily frightened, and they had spells of fainting and unconsciousness of about five minutes' duration. At no time have they had convulsions, or bitten their tongues; that is, there has been no evidence of epilepsy. Both had crying spells, for hours, for no apparent reason. Gradual development of a lack of muscular coordination was observed. Twin *X* developed "a peculiar dragging walk"; she seemed to "walk from her knees down." Twin *Y* also showed incoordination, though less pronounced. Both twins had measles in infancy; both underwent tonsilectomy in 1930. Abnormally high instens (*pes-cavus*) and distended abdomens characterized both twins from infancy.

At the age of 3-½ years, twin *X* was admitted to the diabetic clinic of the Cedars of Lebanon Hospital. Glycosuria had been noted in a previous examination. At this time she suffered an acute and severe onset of diabetes; outstanding symptoms were anorexia, thirst, loss of weight and strength. She was

TABLE II. Oscillometric readings* (Collens oscillometer) by index.

		Normal Range	Mother of Twins	Twin *X*	Twin *Y*
Mid arm:	Right	5 - 12	3	1.25	1.5
	Left	5 - 12	3	1	1.
Fore arm:	Right	3 - 10	2	0.75	1.25
	Left	3 - 10	2.5	1	1.25
Mid thigh:	Right	8 - 15	2	1	1.5
	Left	8 - 15	2	1.25	1.5
Mid leg:	Right	4 - 8	2	0.75	2
	Left	4 - 8	2	2	1.5

*An interaction of neuro-muscular weakness with the peripheral vascular disease may be responsible for these extremely abnormal oscillometeric readings.

TASTE BLIND DIABETIC TWINS

Figure 3

Diabetic symptoms were noted in one twin at three and a half years of age. The other twin, then normal, developed severe diabetes less than half a year later. The twins also suffer from a syndrome of neurological symptoms apparently inherited from their mother.

put on a diabetic diet and treated with insulin.

At the time that severe diabetes was detected in her twin, *Y* was subjected to routine examination and occasional glycosuria was found although her blood sugar was normal. Five months later, twin *Y* developed hyperglycemia, frequent urination, diabetic coma, and ketosis. She was put on a diabetic diet and treated with insulin.

Later Development of the Twins

Since their admission to the Diabetic Clinic of the Cedars of Lebanon Hospital eighteen years ago, the twins have been under frequent observation, and authentic records, rich in detail, are on file. There is little reason, in this paper, to discuss or even to present all of this information, and only a brief survey will be attempted here.

Their diabetes was the primary reason for the detailed medical attention given the twins. Perhaps related to the diabetes has been the peripheral vascular syndrome developed by both. This disease entity is caused by an ischemia of the arterial blood flow in the extremities. It is objectively demonstrated by the Collens oscillometric readings summarized in Table II. The syndrome is most extreme in twin *X*. For readers unfamiliar with the expression of this kind of disorder, a typical record on twin *X* may be quoted in part: "September 10, 1947. Twin *X* complains that her legs hurt her from the hips all the way down;

she cannot touch them at all. Cramps come on after walking for about 3 blocks. Rest relieves cramps. Examination shows marked rubor of legs on dependency; no blanching on elevation. Both hands and feet are rather cold; edema of both legs present. Pulsations of arterial dorsalis pedis and posterior tibialis are diminished."

Peripheral vascular disorders are relatively common among people who have had diabetes over a long period of time. It is therefore not surprising to find this syndrome in the twins. The appearance of the same sort of abnormality in the mother, however, suggests at least some genetic predisposition to this condition in the twins, independent of their diabetes. The mother has never shown evidence of diabetes.

Another abnormal condition, presumably independent of the diabetes and the peripheral vascular disease, found in both twins and similar though less extreme in their mother, is a definite neurological lesion. The diagnosis is based on criteria comparable to those listed for the mother; clinical evidence on neurological examination showing tremor of tongue, markedly impaired gait, ataxia and incoordination of upper and lower extremities. Abnormal deep reflexes and absence of superficial abdominal reflexes as well as presence of the Babinski sign are notable. The details of the diagnosis on many occasions of a diffuse lesion of the brain will not be discussed here. This syndrome is more severe in twin *X*.

As previously noted, the mother gives a normal electro-encephalographic record. The report of Dr. Nicholas A. Bercel, Director of the Department of Encephalography of the Cedars of Lebanon Hospital, may be quoted to summarize the abnormalities of the twins in this respect:

Twin *X*—The routine record shows a dominant frequency of 16 c/s at 40 u v. A moderate amount of 4 - 5 c/s activity at 50 u v is noted in both pre-frontal and occipital areas. Overventilation increases the frontal showing. Impression: This is an abnormal record. It is too fast in general and there is additional slow wave activity in front and in the back.

Twin *Y*—The routine record shows a dominant frequency of 14 - 16 c/s at 30 - 50 u v. Bifrontally several runs of 5 - 6 c/s activity are seen, which at times extends to the parietal area. Overventilation shows no change. Impression: This, too, is a faster than normal record with additional slowing bifrontally; but here the occipital areas are not involved. This is an abnormal record.

The final abnormality to be mentioned is that of mental deficiency or retardation. The twins were early placed in a special class at school. Several of the clinical records refer in a rather general way to the retarded mental development of both twins; to their "childlike" or "rather infantile" speech; to their difficulty with the most simple arithmetic, for example, in an examination at fifteen, twin *X* "knows that she has brothers but does not know if they are older than she is."

Summary and Conclusions

At least five distinctive (though not necessarily independent) abnormalities are present in both these identical twins: (1) Diabetes, noted in each between three and four years of age; somewhat milder in one than in the other. (2) Peripheral vascular disease of upper and lower extremities, alike, though somewhat different in severity, in both twins. (3) The development of a rather bizarre neurological lession of similar nature and simultaneously in both twins. (4) Mental deficiency, alike in the twins. (5) Abnormally high insteps (*pes cavus*). The detailed resemblances of these two individuals, coincident with their identical genetic constitutions, serves to emphasize the significance of heredity in human variation. The fact that certain of these abnormailties are also found in the mother of the twins, in comparable though less severe form, may also be of considerable genetic interest.

It would appear that the parents of the twins are both heterozygous tasters for PTC. It may of course be only coincidence that diabetes and inability to taste this compound occur together in their identical twin children, while the non-diabetic brothers of the twins are tasters. The case is, however, consistent with the already-established,[1] significant

association between diabetes and taste-blindness to PTC in human populations, and something more than coincidence may therefore be involved.

The precise physiological or genetic basis for the high frequency of PTC non-tasters among diabetics remains unknown. The pertinent observation here may be that these diabetic twins, taste-blind to PTC are *tasters* for brucine. This may suggest, though it certainly does not prove, that non-tasting in diabetes is specific for PTC and is not a "lessening of taste acuity generally.[3] On the other hand, the delayed reaction given by the twins, when compared with normal brucine tasters, may support the opposite conclusion: that there is a general lessening of taste acuity in these diabetic twins. Further work with brucine, both in normal and in diabetic subjects, is indicated.

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MORE "BLACK BABIES"

To the Editor:

THE "black baby" has played too large a part both in literature and folk-lore to be easily explained away as a product of the imagination or a result of race prejudice. Probably the basis is not the same in all cases. Some 32 years ago a case was publicized in St. Louis newspapers, in which a white man married a woman who explained her rather dark complexion as due to "Spanish descent." Later a "black baby" was born, the father applied for a divorce and the mother was alleged to have some Negro ancestry. This appears to be fairly typical of such reports. As white infants have broad, flat noses, a diluted amount of such Negro characters as skin color or kinky hair may give an infant a more definitely negroid appearance than it would have in later life.

In the very shadowy case of a "black child" cited by Curt Stern,* he was never able to get nearer to the facts than hearsay statements of a relative concerning events which happened 50 years ago. As it has been estimated that some 10,000 pass for white every year in this country, white persons with some colored ancestry must be more frequent than is generally realized.

To the average person without biological or anthropological training the "black baby" would be a general impression, not depending on skin color alone but probably derived equally from indications of a broad nose and thick lips as well as kinkiness of the hair. Among adults, a racial character of the Negro to which little attention has been given is the very small ear without a lobe. It is also typically flat and close to the head, often with a broad helix (much folded over). That it is highly specialized in regressive evolution is shown further by the fact that Darwin's tubercle is generally absent or small, although the satyr tubercle is frequently well-marked. The evidence also indicates that the small lobeless ear of the Negro is dominant in crosses with whites, and is thus frequently found in those of mixed descent.

The object of this note is to put on record what is believed to be the first case in which a "black baby" and the mother came under scientific observation. It was encountered during the study of a number of colored families in a particular neighborhood in Nova Scotia where there is a considerable colony descended from slaves who went with Loyalist families from the American colonies during the Revolution. One of these families is said locally to have had as many as 32 slaves. They were all set free early in the nineteenth century. Many of the descendants are now so mixed with white blood that some of their offspring can and do pass for white in other parts of the country. Every sort of combination of the racial characters can be found in this settlement, the colored element constituting 18% of its population in the census of 1941.

From information received, I was able to visit the family concerned and obtain full information, but of course, names cannot be given. The wife's family were French Huguenots whose ancestors came to Nova Scotia about 1750. The wife, whom I interviewed, lived in a good house with her father and son (the "black baby"). Her father, who was seen, was unmistakably negroid, but was far from being pure Negro, the kinky white hair being the most marked negroid feature. His wife was dead, but I was told that she was a white

*Stern, Curt. The skin color of children from white by near-white marriages. *Jour. Hered.* 38:233-234. 1947.

woman from another part of the province. There were two sons and a daughter.

The daughter (i.e. the mother of the "black baby") was examined for negroid characters as carefully as circumstances would permit. She was a rather small woman perhaps 45 years of age. She had thin features and a peculiar slightly reddish tint to the face, which is not infrequently seen in segregates from such crosses when the melanin pigmentation is absent. To the ordinary observer she could pass for white. She was wearing no stockings and the skin of her bare legs was indistinguishable from that of a white person. She had brown eyes; her hair was definitely dark reddish brown and curly. Her nose and lips showed no negroid feature. The type of curl of the hair and the peculiar complexion were the only features that in any way suggested Negro ancestry.

I was shown photographs of her two brothers, one of whom had been drowned. As far as could be judged from these cabinet photographs, they were of even purer white phenotype than their sister. Judging from his photograph, her husband also would be regarded as of pure white descent, and this appeared to be his reputation in the village. He was of German and Scotch ancestry, with a German surname. As will appear, however, there is reason to doubt the complete absence of Negro blood from his ancestry. His sister, whose photo I was shown, passes for white and has two children. Her eyes and hair were stated to be brown, the skin and features appeared white in the photograph, but the lips were a little full. Moreover, her hair was wavy with a natural curl, and her sister-in-law stated that she "never requires a perm." This appears to be the type of curl appearing in some Negro-white crosses, which Lotsy called "permanent wave."

If this woman had a small amount of Negro ancestry, as appears from the evidence, then her brother (the father of the "black baby") also had, although there was no indication of it in his photograph. Unfortunately he left the wife after the "black baby" was born, and so could not be seen.

This couple had two children. The eldest was a daughter 22 years old. She passed for white and was a nurse, married to a white man who was a medical student.

The second child was the "black baby." He was 18 years old when seen, had received his "B" certificate in high school and was studying certain subjects in the "A" (12th) grade. He was tall, 5 ft. 9 in., considerably taller than his mother. His skin color was a pale olive, but he could pass for sallow white were it not for his other features. His eyes were dark brown, hair near black, somewhat kinky; ears rather small, with no lobe. His nose was decidedly broad and lips rather thick, these two features making him a "Negro" to the ordinary observer.

Thus the slightly kinky hair together with the nose and lip characters gave him a negroid appearance and made him a "black baby." When he was born the father left, either assuming that he was not the real father or being disgusted at the negroid turn which Nature had taken. He was generally supposed to be of pure white ancestry, but the characters already mentioned in his sister suggest that he, too, probably had a Negro of mixed ancestry somewhere in his ancestry.

Of course, there is no proof that the son was not an illegitimate child. But on the other hand, the reputation of the mother and the opinion in the village was opposed to this interpretation. There is no doubt that the son looks more negroid than his mother or sister, but this is quite in harmony with the genetics of such crosses so far as we know them. The presumption is that we have here a true case.

The current theory of skin color inheritance requires revision. A work now in press* will show that the skin color factors are not only cumulative but unequal in effect. It is evident that some children of colored couples should be darker and some lighter than either parent. Colored families themselves say that some of their children have a darker skin than either parent. But the present observations suggest that other characters than skin color have played a part in producing the widespread impression of a "black baby."

I was told about an even more striking case of a "black baby" which appeared in a very respectable family, but as all information about the people concerned was refused, the case cannot be investigated.

R. Ruggles Gates

Biological Laboratories
Harvard University

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Students of human genetics will note with special interest the following points in Dr. Gates' communication:

1. "The black baby has played too large a part both in literature and folk lore to be easily explained away as a product of the imagination or a result of race prejudice." Also well attested in literature and folk lore are such beliefs as maternal impressions, werewolves, telegony, vampires, and the power of the moon to make or mar the farmer's crops. Despite this imposing weight of literary and folklore "evidence" all of these superstitions are today relegated to the limbo of old wives'

*Gates, R. Ruggles. *Genetics of Negro Families.* 1948. (In press.)

tales. This reason for perpetuating the black baby folk-tale seems utterly fallacious.

2. Dr. Stern's case was not "shadowy" in the sense that is here implied. The recurring story of the "black baby" with which Stern was regaled always contained certain definite and specific details. These details gave the story its main impression of authenticity. Folk-yarns often parallel each other remarkably, as does Dr. Stern's case and the St. Louis case cited favorably by Dr. Gates. By interviewing members of the family involved, Dr. Stern established definitely that these convincing circumstantial details were definitely not true. The child was not black. There *was* a divorce, but the child went with the father. The oft-cited twins did not exist. The statement by a competent witness able to give definite information on these points is not "shadowy" and it is relevant to the matter in hand. The fact that these folk-tales abound with highly circumstantial supporting details which cannot be verified is one of their interesting and well-recognized anthropological features. Such details hardly validate the rest of the yarn.

As a matter of fact, Dr. Stern's information seems a great deal more substantial than that which Professor Gates reports in this "first case in which a 'black baby' and the mother came under scientific observation." The observations Professor Gates reports seem hardly conclusive. The case hinges on whether or not the husband had any negro blood. And on this point this "scientific record" is admittedly not positive. Cabinet photographs of long deceased individuals are hardly acceptable as a basis for reaching final conclusions. And as Professor Gates admits, there is a possibility of illegitimacy. So that this first scientific case to be reported is at best "only by presumption" an actual instance of a "black baby," and not so very black at that!

3. As pointed out by Stern, the present genetic concept of inheritance of human color differences assumes that there are at least two pairs of alleles, cumulative in their effects. This means that if a child is blacker than either parent the genes for heavy pigmentation must be carried by both parents, so that the number of "plus genes" can be increased in the child. Hence this case, even if taken at face value seems wholly inadequate to require that "the current theory of skin color inheritances requires revision."

The case reported by Professor Gates seems somewhat less striking and scarcely better grounded than the reference submitted by a correspondent, and published in the JOURNAL OF HEREDITY shortly after Stern's report. And if literature and folk lore are to be accepted at face value in studies of human genetics, why not give full faith and credence to the reports of such distinguished literary authorities as Dr. Watson and Mr. Sherlock Holmes, and in real earnest add them to the "scientific literature" of the subject?—EDITOR.

Eugenic Significance of Multiple Factors

FROM the point of view of practical eugenics, the kind of hereditary difference which is determined by multifactorial inheritance is far more important than that caused by single genes. Here are some of the reasons. First, those things determined, or partly determined, by multifactorial inheritance are more important socially. Intelligence, good physique, resistance to disease are more important to the community than rare hereditary diseases, even including some of the low-grade mental deficiencies, or those diseases which though rather commoner appear to have a definite and maybe simple genetic basis. In the same way, temperamental differences are more important than the insanities. All the hereditary mental illnesses together give a risk of only about 2 per cent in the general population, whereas the disabilities caused by neurosis and psychopathy are of fateful significance to us all.

Secondly, and this is even more important, simply inherited abnormalities are individually rare or very rare. Seldom indeed does a hereditary defect attain a frequency of one in 5,000 in the population; it is usually much less than this. The sole exception is colour-blindness, but this condition should perhaps be regarded as a more or less physiological variation rather than as an abnormality. Selection then, either natural selection based on natural differing rates of reproduction, or any conscious selection that it might be decided to apply, operates on a few human being only. With recessive genes it is only very occasionally that we can identify genetically the outwardly normal carriers, so our general conclusion is little affected. Contrast with this multifactorial inheritance, determining in part some human quality such as general intelligence. Every human being exhibits the result of the action of many genes, some good, some bad. With the birth of every single human child the balance tips a little, up or down. Selection is operating, not on one person in five thousand, or fifty thousand, or five hundred thousand, but on every child born to every couple; for upon the sum total of their good and bad genes and the relative rates at which these are increasing or diminishing depends the future of the species, whether it is advancing, whether it is holding its own or whether it is retrogressing. Here is something about which the individual can have his say. Here is something not merely about which the State can legislate effectively if it wishes, but something which is automatically affected, for better or for worse, by social and economic measures of many kinds.—J. A. FRASER ROBERTS and ELIOT SLATER—*Eugenics Review*, July 1948.

CHIMERAL APPLE SPORTS

And Their Propagation Through Adventitious Buds

HAIG DERMEN*

A STUDY of chromosomal chimeras as induced by colchicine treatment has broadened our knowledge of the developmental morphology of the growing stem- and root-tips of plants. The work of Satina, Blakeslee, and Avery,[8, 9, 10] and of the author[2, 3, 4] has established the fact that in stem growth of perhaps all angiosperms three distinct histogenic layers of cells are involved. This is in contrast to the situation in gymnosperms, where no layering of cells takes place. As a consequence one would expect to find a number of patterns of periclinal chimeras in angiosperms which would not be found in gymnosperms. In the latter group sectorial chimeras extending all the way from bark to pith would be expected, but no periclinal chimeras. In the angiosperms, on the other hand, chimeras involving any one or more of the three primary cell layers would be expected. This distinction with respect to chimeral types encountered in these two classes of plants appears to be borne out by an analysis of chimeras observed in them.

This concept has important implications in the utilization of chimeras for theoretical researches in plant histology and in applied research in horticulture. It will help us to understand the sometimes paradoxical results reported in colchicine experiments. It should enable us to achieve greater precision in the use of colchicine to produce polyploid forms for horticultural or genetic purposes.

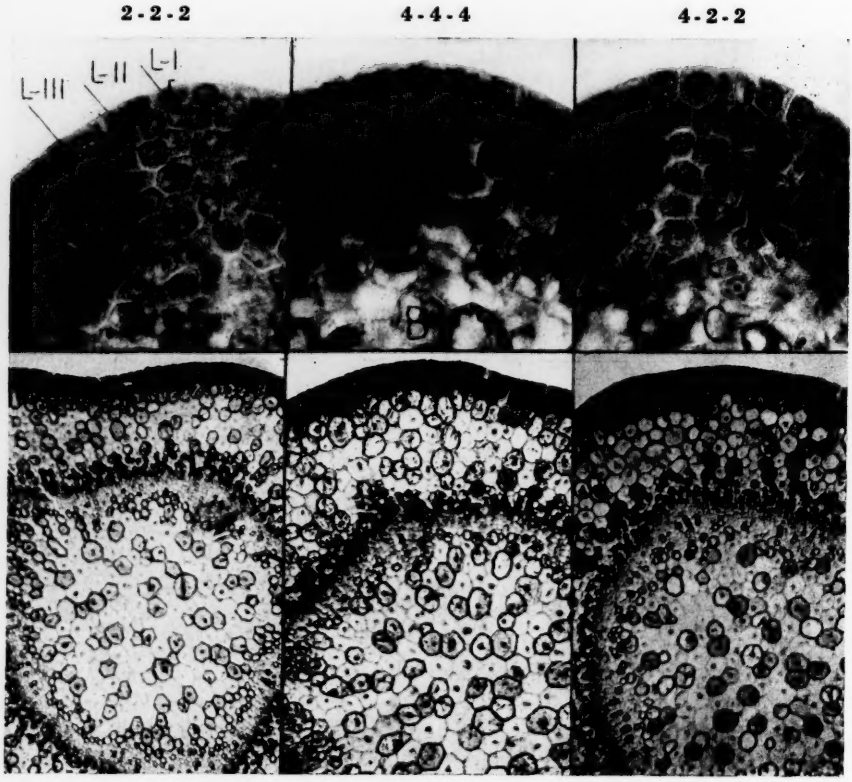
Three Layers

The three primary histogenic layers have been labelled L-I, L-II, and L-III. Each layer remains quite distinct from the others at the shoot apex. This is due to the fact that cell division in the two outermost layers, L-I and L-II, is usually in an anticlinal plane; that is, at right angles to the surface of the apical dome. Thus these layers remain quite distinct as single layers of cells at and immediately below the surface of the apical dome. The plane of cell division in the innermost, third, layer (L-III) is both periclinal and anticlinal with respect to the surface of the apical dome. Hence in contrast to the other two layers, this layer (L-III) is responsible for the growth in the shoot apex in depth as well as in extent (Figure 4).

In general, the histological structure in the stem back of the apical dome in horticultural plants is as follows: the epidermis of the stem, leaf, and fruit is derived from the outer histogenic layer (L-I). The intermediate histogenic layer (L-II) behaves differently in different species and in different organs. In the cranberry stem it gives rise to a one cell layer thick hypodermal tissue but in the peach it gives rise to an outer cortex tissue that is many cells thick lying just under the single layer of L-I cells. It is also the source of tissues in the cranberry fruit of a varying number of cells in thickness. In the fruit of both the cranberry and the peach there is an L-II tissue several cell-layers thick situated next to the epidermal tissue (L-I) which lines the ovarian cavity of the fruit. The remaining tissues in both the stem and the fruit are believed to be derived exclusively from L-III.

Since throughout the development of the plant these layers usually remain distinct, and independent of each other, a mutation occurring in a shoot apex will

*Cytologist, United States Department of Agriculture, Agricultural Research Administration, Bureau of Plant Industry, Soils, and Agricultural Engineering, Division of Fruit and Vegetable Crops and Diseases, Plant Industry Station, Beltsville, Maryland.



PLOIDY IN THE THREE GROWTH LAYERS

Figure 4

The upper row of photomicrographs shows longitudinal sections through the growing tips of cranberry shoots. The three histogenic layers are designated in the left-hand photograph (*A*), and the ploidy of the three layers is shown in the numerical formulae above the photographs. The segment shown at *A* is composed of exclusively diploid tissue in all three layers (formula: 2-2-2). *B* shows sections of a completely tetraploid shoot and stem, wherein all three layers are 4*x* (formula: 4-4-4). *C* shows a chimera in which the outer histogenic layer (L-I) is tetraploid and the two inner layers are diploid (formula: 4-2-2). In the stem a single outer layer of cells is composed of L-I tissue; the next layer is, like the growing point, composed of L-II tissue; and all of the interior of the stem is composed of L-III tissue. Differences in cell size in diploid and tetraploid tissue are more marked in the internal parts of the stem than in the L-I and L-II layers.

be localized, at least initially, to only one of the three layers. That is true whether the "mutation" is a gene mutation involving a specific chromosome locus, or a mutation in ploidy involving a multiplication or reduction in the number of chromosomes. For this reason, sports resulting from genic or chromosomal changes in angiosperms are almost always found to be periclinal chimeras. The "sport" is genetically or cytologically different from the remainder of the original plant which is unaffected by the change in the chimeral zone. It is important that the chimeral nature of most sports be recognized, because it is possible to convert these from their initial chimeral condition to a branch or

plant homogeneous with respect to the "mutant" form. The means whereby such conversion to homogeneity can be achieved is the subject of this paper.

Layer Replacement

In our studies of the cranberry and peach, no evidence was produced that any internal tissue in the stem, leaf or fruit is derived from the L-I layer except the thin L-I lining of the ovarian cavity. However, there was clear evidence from the study of varigation in certain other plants[4] that in some cases the L-I layer does give rise to internal tissue as well as to epidermis. Occasionally, it appears that in these other plants the original L-II is replaced by a layer of cells originating from L-I through a periclinal cell division of this latter layer. As a result of such replacement, L-III tissue is sometimes replaced by L-II tissue. When this happens L-III can be completely lost from the apical meristem so that it takes no further part in apical growth. Our technique for making certain chimeras into homogeneous plants depends upon the process of such replacement of one histogenic layer by another.

Replacement of the original L-III by a daughter layer from L-II may take place at least in some plants more often than replacement of L-II by L-I. When a replacement is partial, the changes may be sectorial (mericlinal) and involve areas of various shapes and dimensions. Such replacements may become the basis either of reversion from sport to normal or of a mutation from normal to sport.

Differential Ploidy and Fruit Size

In addition to the possibilities above indicated, the number of cells derived from L-II and L-III during the development and growth of the fruit may be variable. Variability in these layers may have an effect on the size of the fruit in cases where the sport is of diploid-tetraploid constitution with respect to L-II and L-III layers. This was indicated in fruits of a cyto-chimeral cranberry[3] where L-I and L-II were $2x$ and L-III was $4x$. In three very young fruits of diploid-tetraploid chimeral cranberry, the following groups of cells* — each traced to a particular layer—were found: In one fruit there were *five* layers of $2x$ cells from L-II under the outer (L-I) epidermis, *three* medial layers of $4x$ cells from L-III, and *seven* layers of $2x$ cells from L-II next to the inner epidermis, lining the seed cavity.

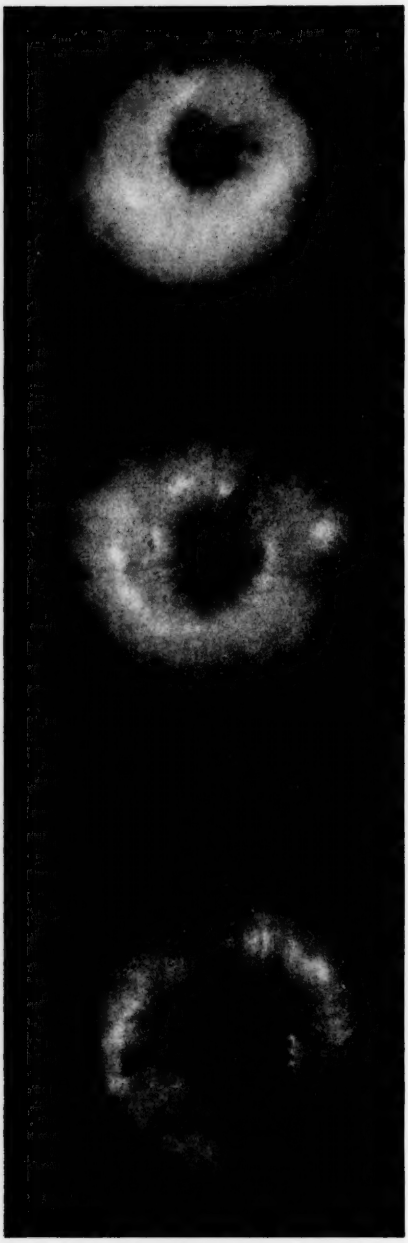
In a second fruit, in the same order of arrangement and ontogeny, there were *nine* layers of $2x$ cells, *six* of $4x$, and *one* of $2x$ cells. In the third fruit, *two* layers of $2x$ cells, *eleven* of $4x$ cells and *three* of $2x$ cells. This illustrates clearly that in fruit development, as in stem and leaves,[3,4] the amount of tissue derived from L-II and from L-III may vary greatly.

Chimeral Situation in the Apple

In the apple, the findings with respect to the chimeral nature of polyploid sports has been the subject of intensive study. It is definitely established by Einset and his co-workers[6,7] and by the Beltsville research team with which this author is associated,[1,5] that in general three distinct chimeral conditions exist depending on the origin of the mutant tissue.

On the assumption that tetraploidy in the apple increases size of fruit, maximum size due to tetraploidy should occur when every tissue of a diploid fruit has become tetraploid. Since only a single cell-layer of epidermis is normally derived from L-I, the fruit size in periclinal chimeras with diploid L-I tissue and the rest of the fruit tetraploid may be virtually as large as in a totally tetraploid fruit. When either the L-II or the L-III layer is tetraploid, the size of fruit may range between that of totally diploid fruit and of totally tetraploid fruit. The fruit size would depend on the relative amounts of L-II and of L-III tissue. In plants having diploid L-II tissue and tetraploid L-III tissue the size of fruits might average larger than in cases where L-II is tetraploid and L-III is diploid. This is due to the number of layers of

*Cell counts were made from only one half of each fruit.



THE "SWEET AND SOUR" SPORT

Figure 5

cells derived from L-II as well as to the fact that they are flattened next to the epidermis (external and internal) and are not fully enlarged, whereas the cells from L-III, being medially located, are enlarged to the fullest possible extent.

Nature of Fruit Color of Sports

The pigment in a colored apple is normally present both in the epidermal layer and to varying depths in the subepidermal tissue. The presence of pigment in the epidermal cells can be detected when fruits are examined before full maturity. At maturity, pigment is generally lacking in the epidermal cells, probably because of protoplasmic deterioration in those cells at a late stage of fruit development. The coloration at maturity of the fruit is due entirely to the presence of pigment in the subepidermal cells.

A mutation affecting color of fruits, of course, may appear in any one of the three histogenic layers of the apple. A color change in L-I, because limited ordinarily to the epidermis, may not be apparent at maturity. The presence of a color sport in L-I tissue is usually detected only if L-I gives rise to some internal tissue in parts of the fruit. In rare instances intense coloration appearing sectorially on some fruits, otherwise normally colored, may be taken as due to the presence of a factor for a sport to intense color in the L-I layer.

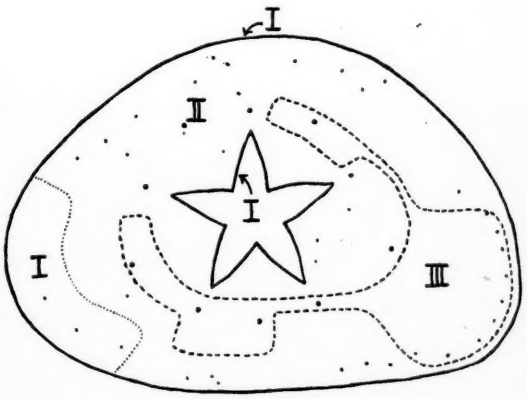
On the skin of sports with intense colored fruits sharply delimited dull-color sectors have occasionally been observed. In such cases it would seem that such a sectoring occurs when, as stated above, the subepidermal tissue is in part derived from L-I. The intense-color factor present in L-II has been replaced by normal colored tissue derived from L-I.

If such replacement of tissues can

A represents the sweet-tasting, small-sized, round fruit of a sport from the "Sweet and Sour" variety. *B* represents the irregular-shaped, medium-sized fruit, in parts sweet and in parts sour, of the "Sweet and Sour" variety, a sport of Rhode Island Greening. *C* represents the normal sour, large, round fruit of Rhode Island Greening.

CROSS-SECTION OF CHIMERAL APPLE

Figure 6



Diagrammatic presentation at a median transection of the fruit of the "Sweet and Sour" variety. I, II, and III designate the tissues or tissue regions presumably derived from histogenic layers L-I, L-II, and L-III, respectively, in this variety. The outer line of the diagram indicates the outer epidermis, and the star-shaped figure in the center outlines the inner epidermis, both derived from L-I. A small portion of the fruit on the left, marked I and delimited internally by a dotted line indicates the possibility that a portion of the internal tissue may, at points where the fruit bulges out, be derived from L-I. A zone in the fruit that may be derived from L-III is shown delimited by a broken line. The rest of the internal tissue may have been derived from L-II. Large dots in the figure represent transections of major bundles going into the sepals and petals of the apple flower, and the small dots represent transections of vascular bundles terminating in the fruit itself. The area on the left side in the diagram marked I and the inner area marked III would represent sour-tasting regions of the fruit, and the remainder, marked II, would represent sweet-tasting regions. On the whole, the bulged portions, at least peripherally, tasted sour in actual tasting tests, whereas the portions of the fruit with normal curvature tasted sweet.

change coloring, it would appear that a color mutation occurring in L-III would never be known, since the tissue or zone in the fruit derived from L-III would almost always be too deep in the fruit to be externally apparent. A color factor in L-III would be detected only if tissue formation in the fruit from L-I and L-II was extremely limited. The presence of a color mutation in L-III would be revealed if shoots were developed from a root cutting or were formed adventitiously from the stem.

The "Sweet and Sour" Sport

An old pomological curiosity, the "Sweet and Sour" sport of Rhode Island Greening, is presented here as an example of a periclinally constituted sport. The fruit of the Rhode Island Greening, shown in Figure 5*C*, is large, round, and decidedly tart in flavor. The fruits of the "Sweet and Sour" sport are mostly irregular in shape, as shown in Figure 5*B*, some parts bulging out, other parts being more evenly rounded or regular in curvature. The sour portion is mostly found in the bulgy part, especially in the flesh near the skin; and the more evenly rounded parts mostly taste sweet. Sometimes this situation seems to be reversed; there is a suggestion of sweet taste in the bulged parts and of sour taste in the regular parts. Not all fruits on a tree of this sport are irregular in shape and mixed in flavor; some fruits taste entirely sweet. Completely sweet-tasting fruits are much smaller than the normal fruit of Rhode Island Greening. They are perfectly shaped without bulging in any portion, as shown in Figure 5*A*. Bud propagations from branches bearing fruits of the latter type have resulted in trees bearing only small, rounded, and entirely sweet fruits.

The diagram from a cross section of "Sweet and Sour" fruit in Figure 6 is based on the account given above. Since it appears that some fruits of the "Sweet and Sour" variety are small, rounded, and sweet, then it may be assumed that a mutation bringing about such changes in the normally large, round, and sour

Rhode Island Greening had occurred in L-II. As was shown in the cranberry, it occasionally happens that some buds develop exclusively from L-I and L-II tissue. Since L-I normally gives rise only to the epidermis, such buds or fruits would have internal tissue derived exclusively from L-II. Therefore, the small size and sweet taste of some fruits would indicate that it is the L-II that carries the mutation conditioning the change in size and flavor of the fruit of the Rhode Island Greening. In the "Sweet and Sour" variety, localization of the sour taste in the outermost portion of the fruit may be explained by assuming that some of the internal tissue was derived from L-I (left portion of Figure 6), or that L-II tissue growth in that region was very limited (right portion of Figure 6). In this interpretation the sour taste would be due to tissue derived mostly from L-III.

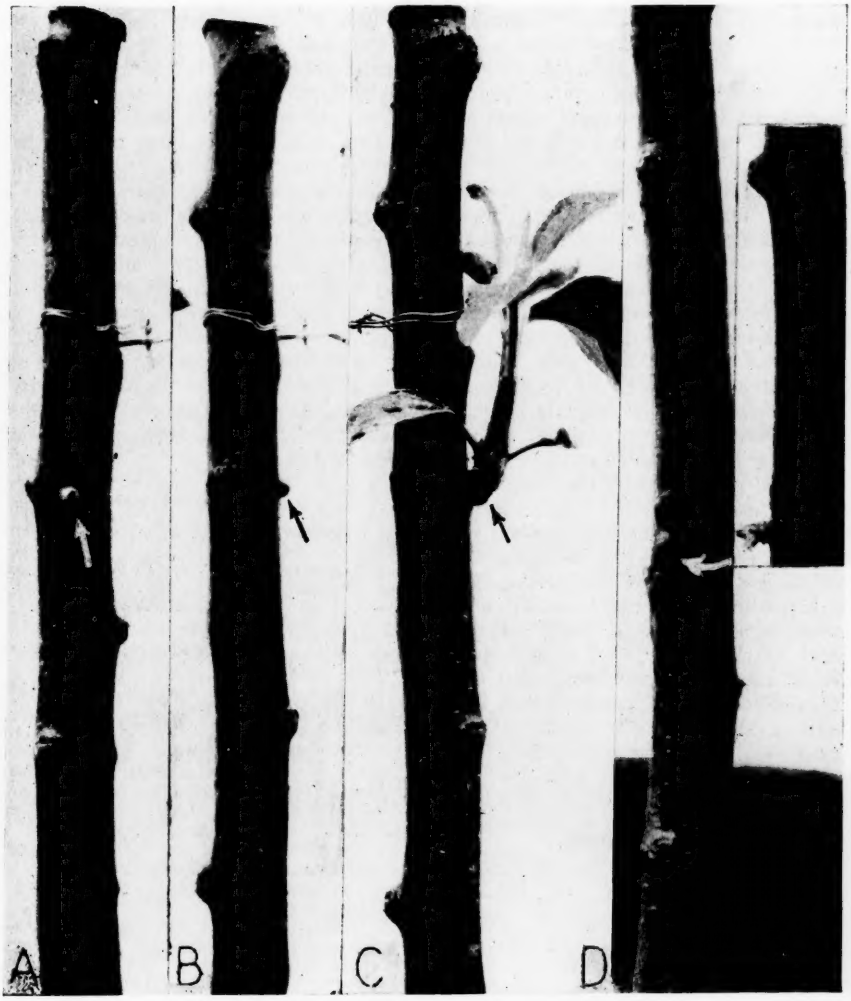
From "Sweet and Sour" no reversion to the Rhode Island Greening type has been observed. This should be so, since in the normal processes of bud and fruit formation L-III can never replace L-II. There is a remote possibility that internal tissue may at times develop from L-I, so that buds with the original Rhode Island Greening characteristics actually may in rare instances develop from the "Sweet and Sour" variety. A reversion from "Sweet and Sour" to the true Rhode Island Greening would most likely occur through adventitious bud development, as has been indicated above. Results from root propagation would be similar to those obtained through an adventitious bud, since both originate in tissue usually derived from L-III.

Adventitious Buds to Order

Some method is needed to obtain a homogeneous tree from one having a desirable sport confined exclusively to the third layer. One way seems to be to obtain adventitious buds and shoots from such a chimeral sport. Adventitious bud growth has been obtained in apple trees by the following method. On February 28, 1946, in preparation for polyploidy work with colchicine, a dozen two-year-old trees of the Lady variety were potted in 12-inch pots and were trimmed back to the main trunk of the trees and topped about two feet above the soil level. In a week or two after the trees were cut back all but two showed new growth from axillary buds. On one of these two trees, by May 7 a small swelling appeared *in an internodal region* of the old stem; this slowly broke through the bark and developed into a shoot. A similar growth occurred much later on the second of these trees, but this failed to develop into a shoot. This authentic case of adventitious shoot development on the stem of an apple suggested a possible way of obtaining adventitious buds which might yield varietal apple sports in a homogeneous state.

On the premise that sports are likely to be periclinally constituted, a number of one-year-old trees of color sports of standard varieties were placed in 12-inch pots and cut back to one foot above the soil level. Since in the case of the Lady variety the adventitious bud development seemed to be associated with lack of growth from buds at the nodes, *all the buds on the young trees were dug out,* in the hope that adventitious bud growth might thus be similarly forced. Soon after disbudding, small buds began to sprout from some of the scars, but since they might conceivably have been derived from basilary primordial buds, they were rubbed off. An almost continuous shoot development took place from the roots of the disbudded trees. These, of understock origin, were constantly destroyed at the point of their development on the roots.

Trees for this experiment, all one year old, were planted at three different times (not intentionally, but of necessity—because of the availability of these trees at different times). The first planting was made on January 20, 1947. The varieties used at this time were Imperial All Red McIntosh (four trees purchased from a commercial nursery) and No. 66 Stayman Winesap (four trees from the nursery at this Station). The second



FROM ADVENTITIOUS BUD TO SHOOT

Figure 7

Adventitious buds on apple stem of a Red McIntosh Apple tree. The white arrow in *A* points to an adventitious bud. The same bud is shown in side view in *B*. In *C* is shown the shoot developed from the adventitious bud shown in the figures on the left. In *D* is shown the basal portion of the same tree. In the area marked by a white arrow there are three adventitious buds. The arrow points to the bud from which the small shoot shown just to the right developed later.

planting was made on April 3, 1947. The varieties used were Jonared Jonathan and Starking (six of each from a commercial nursery). The third planting was made on April 15, 1947. The varieties used at this time were again No. 66

Stayman Winesap (eight trees from this Station) and Scarlet Red Stayman (ten trees from a commercial nursery). The plants were kept in the greenhouse until August 10, 1947, when they were moved outdoors into a screen house for overwintering.

Adventitious buds like those shown in Figure 7 developed quite readily on all Red McIntosh and Starking trees and sparsely on Jonared and the two Stayman Winesap strains. Shoots developed from adventitious buds on all McIntosh and Starking trees; but only one shoot developed on a Stayman tree, this last development, involving the No. 66 strain, taking place in late August, after the plants were moved outdoors.

The adventitious nature of these buds could be recognized by their highly abnormal growth behavior: (1) the buds occurred in the internodal region of the stem of young trees, where normal buds are absent; (2) they were found being initiated in the outer phloem; (3) at the point of emergence of such bodies the bark cracked open; (4) there were no foliar appendages on these bodies when they made their appearance and for a considerable time afterwards; (5) the growth of these bodies and their development was very slow; (6) often from one body more than one shoot developed.

Conclusions

It appears that adventitious buds may develop quite readily on some varieties through use of the method here described, and on others with difficulty, or perhaps not at all. Such attempts may have to be made under different environmental conditions—outdoors as well as in the greenhouse—to test the possibility of success with certain varieties.

The method of obtaining adventitious shoot development here described may be especially valuable in cases of diploid-tetraploid periclinal chimeras where tetraploidy is found in the innermost tissue, as in the Northern Spy chimeral form reported by Einset *et al.*[6,7] Adventitious shoots from such forms would be expected to be homogeneously tetraploid. From similar chimeral forms homogeneously diploid, adventitious shoots may be obtained if at times the L-III layer were replaced by L-II in the manner described earlier.

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*Through an error this was called a triploid in the original article in the Journal. The Editor regrets.

THE DEFINITIVE HAWKS-BEARD

A Review

PROFESSOR Babcock's two volume masterpiece* is no subject for a timid reviewer. Thirty years of intensive work in genetics, cytology, and taxonomy are summarized in two well-illustrated, complexly documented volumes. A mere catalogue of what is to be found in these two compendia would take up more space than the average review. Nor is that the only difficulty; of the more than a thousand pages of reading matter, some can be read through quickly but there are many which must of necessity be studied carefully.

Take the attractive frontispiece of volume 1 for example. It was carefully conceived as a kind of visual epitome of Professor Babcock's main thesis concerning the course of evolution in *Crepis*. It can be taken in at a glance but it deserves careful study and quiet reflection. Two species of *Crepis* are illustrated in full color with detailed enlargements of seed heads, flower parts, and seeds, along with drawings of metaphase chromosomes. Those who know *Crepis* only as a name will now realize that it is a genus of weedy little plants, rather like coarse, small-flowered dandelions. One of the species illustrated represents one of the most primitive of the five- chromosomed species, the other one of the most advanced. A good part of the volume is given over to the presentation of evidence from various fields, abundantly justifying Babcock's conclusion that the general course of evolution in *Crepis* has been from large-seeded perennial mesophytes to small-seeded annual xerophytes.

Volume 1, a mere 197 pages in length, is (as we learn from its sub-title) devoted to the taxonomy, phylogeny, distribution, and evolution of *Crepis*. An introduction of 27 pages outlines the history of the investigation and the general lines it has taken, with generous acknowledgments to the various individuals and organizations who have been most intimately associated with the program. There is a long discussion of the principles of systematic work in general and of the systematics of *Crepis* in particular, including much information derived from cytological examination and genetic experimentation. To many biologists, already familiar with the general trend of cytogenetic research in *Crepis*, the most interesting sections will be those on geographical distribution of the species of *Crepis* and Asiatic-European plant migration in the Tertiary period. Here the reader will find a correlated discussion of facts from taxonomy, palaeontology and geology which is of central significance to much more than the evolution of the genus *Crepis*.

The facts of endemism in *Crepis* are collated and discussed, Willis's concept of Age and Area being shown to be completely inadequate and misleading. The volume closes with a detailed 12-page discussion of outstanding *Crepis* problems which deserve further investigation.

A monograph of the genus *Crepis* is presented in the 1030 pages of volume 2. There is a line drawing for every one of the species, with detailed close-ups of seeds, flower parts and other significant details. Where the chromosome number has been determined there are drawings of the metaphase chromosomes. There are in addition over 30 plates of photographs of herbarium specimens, an index to generic and specific names and an index to Exsiccatae (taxonomic *patois* for herbarium specimens) which occupies two columns a page for 14 pages. For each of the species there are: a complete technical description, a general discussion of the distribution and

*Babcock, Ernest Brown. *The Genus Crepis.* Two volumes, illustrated. Univ. Cal. Press. $15. 1947. (Part I. *The Taxonomy, Phylogeny, Distribution and Evolution of Crepis.* U. Cal. Pub. Bot. 21(12):1-198. $4.00. Part II. *Systematic Treatment. ibid* 22(10):199-1030. $12.)

relationship of the species, a citation of specimens which have been examined and a discussion of the relationships of that species to the others in the genus.

As one bores into this volume he begins to realize the years of work and the careful systematic planning which have made it possible. Of the 196 species in the genus, 113 have been grown under experimental conditions and examined cytologically. Herbarium specimens have been consulted from 85 different herbaria. During thirty years various American colleagues have been directly associated with Professor Babcock in studying *Crepis;* Stebbins, Jenkins, Hollingshead, Collins and Cameron have all made significant contributions. Four foreign colleagues have worked on the project and 13 graduate students.

During almost the entire course of the investigation the project has been carried on as a piece of basic research in the California Agricultural Experiment Station. Additional funds have been supplied by the Carnegie Institution, the Rockefeller Foundation, the Society of Sigma Xi, and the American Philosophical Society. It is to the lasting credit of these institutions that the project was supported and the volumes have appeared. Particularly noteworthy is the fact that the Experiment Station, through several changes of administration, continued its active support of a large piece of research of no conceivable direct practical value.

Without withholding any of the glory from these institutions, one needs to point out specifically that it was Professor Babcock who from first to last was center of the project. Its completion is a monument not only to his ability as a scientist but to a real genius in human relationships. For these thirty years he has gone so deftly about the complicated business of getting support for his unusual project that even to most of his colleagues it appeared as if the whole concern was just running itself. The greatest art conceals its own mechanics; this unprecedented integration of scientific cooperation did not just happen. It has been apparent to nearly everyone who met him that Professor Babcock was "gentle as a dove"; it is equally true that he has been "as wise as a serpent." Within his own institution and outside it, he has quietly brought about a spirit of cooperation with his program. Somehow, in the midst of a large complex academic machine, the funds and the space and the colleagues, and the critically assembled research materials from all over the world, and the students, and the research time, and the technical assistance, and the mediums of publication have all been brought together.

EDGAR ANDERSON
Missouri Botanical Garden

◆

Department of Aristotelian Understatement

(or putting the cart of addition before the horse of multiplication)

Cereals, including rice, now directly provide something like 75 per cent of all the food eaten by the peoples of the world. Nor is that the extent of their importance, for cereals make still another heavy contribution to the world's food larder through their use as livestock feed.

Dependence on cereals has been accentuated by the failure of *total* food production to keep pace with population growth during and since the war. *Not until total agricultural output increases at a rate in keeping with population growth* will it be possible to restore and increase the relative place of livestock products in the food supply. (Italics editor's.)

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